	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	33	gastric adj proton adj pump adj inhibitor	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:22			0
2	BRS	L2	1419	rabeprazole or omeprazole or lansoprazole or pantoprazole	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:23			0
3	BRS	L3	3114	gastrin or pentagastrin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:24			0
4	BRS	L4	60	(1 or 2) same 3	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:24			0
5	BRS	L5	66	excess adj gastric adj acid adj secretion	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:25			0
6	BRS	L6	3069	(zollinger$1ellison adj syndrome) or (gastroesophageal adj reflux adj disease) or (peptic adj ulcer adj disease) or (atrophic adj gastritis) or esophagitis or (idiopathic adj gastric adj acid adj hypersecretion)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:28			0
7	BRS	L7	10	4 same (5 or 6)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:40			0

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
8	BRS	L8	107330	antibiotic or penicillin or tetracycline or macrolide or cephalosporin or fluoroguinolone	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:42			0
9	BRS	L9	0	7 same 8	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:42			0
10	BRS	L10	0	4 same kit	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/17 14:42			0

> d his

(FILE 'HOME' ENTERED AT 14:44:23 ON 17 NOV 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
14:44:51 ON 17 NOV 2002
L1 185 S GASTRIC PROTON PUMP INHIBITOR
L2 36717 S RABEPRAZOLE OR OMEPRAZOLE OR LANSOPRAZOLE OR PANTOPRAZOLE
L3 81692 S GASTRIN OR PENTAGASTRIN
L4 3158 S (L1 OR L2) (P) L3
L5 89633 S (GASTRIC ACID SECRETION) OR (ZOLLINGER ELLISON SYNDROME) OR (
L6 1066 S L4 (P) L5
L7 418 DUPLICATE REMOVE L6 (648 DUPLICATES REMOVED)
L8 217 S L7 (P) TREAT?
L9 108 S (L1 OR L2) (A) L3
L10 19 S L9 (P) L5
L11 11 DUPLICATE REMOVE L10 (8 DUPLICATES REMOVED)
L12 969962 S ANTIBIOTIC OR PENICILLIN OR TETRACYCLINE OR MACROLIDE OR CEPH
L13 9 S L8 (P) L12
L14 9 S L13 NOT L11
L15 0 S L7 (P) KIT

=> log y

```
FILE 'HOME' ENTERED AT 14:44:23 ON 17 NOV 2002

=> file medline caplus biosis embase scisearch agricola
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 14:44:51 ON 17 NOV 2002

FILE 'CAPLUS' ENTERED AT 14:44:51 ON 17 NOV 2002
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 14:44:51 ON 17 NOV 2002
COPYRIGHT (C) 2002 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'EMBASE' ENTERED AT 14:44:51 ON 17 NOV 2002
COPYRIGHT (C) 2002 Elsevier Science B.V.  All rights reserved.

FILE 'SCISEARCH' ENTERED AT 14:44:51 ON 17 NOV 2002
COPYRIGHT (C) 2002 Institute for Scientific Information (ISI) (R)

FILE 'AGRICOLA' ENTERED AT 14:44:51 ON 17 NOV 2002

=> s gastric proton pump inhibitor
L1          185 GASTRIC PROTON PUMP INHIBITOR

=> s rabeprazole or omeprazole or lansoprazole or pantoprazole
L2        36717 RABEPRAZOLE OR OMEPRAZOLE OR LANSOPRAZOLE OR PANTOPRAZOLE

=> s gastrin or pentagastrin
L3        81692 GASTRIN OR PENTAGASTRIN

=> s (l1 or l2 ) (p) l3
L4         3158 (L1 OR L2 ) (P) L3

=> s (gastric acid secretion) or (zollinger ellison syndrome) or (gastroesophageal reflux disease)
   3 FILES SEARCHED...
L5        89633 (GASTRIC ACID SECRETION) OR (ZOLLINGER ELLISON SYNDROME) OR
                (GASTROESOPHAGEAL REFLUX DISEASE) OR (PEPTIC ULCER DISEASE) OR
                (ATROPHIC GASTRITIS) OR ESOPHAGITIS OR (IDIOPATHIC GASTRIC ACID
                HYPERSECRETION)

=> s l4 (p) l5
L6         1066 L4 (P) L5

=> duplicate remove l6
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L6
L7          418 DUPLICATE REMOVE L6 (648 DUPLICATES REMOVED)

=> d his

     (FILE 'HOME' ENTERED AT 14:44:23 ON 17 NOV 2002)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     14:44:51 ON 17 NOV 2002
L1          185 S GASTRIC PROTON PUMP INHIBITOR
L2        36717 S RABEPRAZOLE OR OMEPRAZOLE OR LANSOPRAZOLE OR PANTOPRAZOLE
L3        81692 S GASTRIN OR PENTAGASTRIN
L4         3158 S (L1 OR L2 ) (P) L3
L5        89633 S (GASTRIC ACID SECRETION) OR (ZOLLINGER ELLISON SYNDROME) OR (
L6         1066 S L4 (P) L5
L7          418 DUPLICATE REMOVE L6 (648 DUPLICATES REMOVED)

=> s l7 (p) treat?
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L54 (P) TREAT?'
```

```
L8          217 L7 (P) TREAT?

=> s (l1 or l2) (a) l3
L9          108 (L1 OR L2) (A) L3

=> s l9 (p) l5
L10          19 L9 (P) L5

=> duplicate remove l10
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L10
L11           11 DUPLICATE REMOVE L10 (8 DUPLICATES REMOVED)

=> d l11 1-11 ibib abs

L11  ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2002:622535  CAPLUS
DOCUMENT NUMBER:          137:179667
TITLE:                    Effect of pantoprazole versus other proton pump
                          inhibitors on 24-hour intragastric pH and basal acid
                          output in Zollinger-Ellison syndrome
AUTHOR(S):                Ramdani, Akli; Mignon, Michel; Samoyeau, Roland
CORPORATE SOURCE:         Service de Gastroenterologie, Groupe Hospitalier
                          Bichat-Claude Bernard, Paris, 75877, Fr.
SOURCE:                   Gastroenterologie Clinique et Biologique (2002),
                          26(4), 355-359
                          CODEN: GCBIDC; ISSN: 0399-8320
PUBLISHER:                Masson Editeur
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   Aim - In this open prospective study, the efficacy of pantoprazole in
     reducing gastric acid secretion in Zollinger-Ellison syndrome patients was
     compared to that obtained previously with other proton pump inhibitors.
     Methods - Eleven male patients previously treated with omeprazole (n = 7,
     mean dosage: 63 mg/day; range: 20-100 mg/day) or lansoprazole (n = 4, mean
     dosage: 75 mg/day; range: 30-120 mg/day) were included.  These patients
     underwent a 24-h intragastric pH-metry, measurement of basal acid output
     and of serum gastrin first while receiving their usual therapy and second
     after 7 to 10 days of pantoprazole treatment at a mean dosage of 116
     mg/day (range: 40-200 mg/day).  Basal acid output was evaluated after each
     intragastric pH-metry, one hour before the next intake of proton pump
     inhibitor and a serum gastrin curve was detd. according to 9 fixed time
     points.  Results - One patient dropped out before the second intragastric
     pH-metry due to an adverse event (varicella) unrelated to pantoprazole and
     was reinvestigated thereafter.  The median 24-h intragastric pH with
     pantoprazole was not significantly different than that with the other
     proton pump inhibitors (5.3 vs. 4.6, resp.; P = 0.90).  Neither the median
     basal acid output values nor the median serum gastrin levels were
     significantly different between pantoprazole and the other proton pump
     inhibitors.  Conclusion - In these patients with the Zollinger-Ellison
     syndrome, pantoprazole was well tolerated and equally effective to the
     other proton pump inhibitors in terms of antisecretory potency.
REFERENCE COUNT:          13    THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11  ANSWER 2 OF 11  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2001:247197  CAPLUS
DOCUMENT NUMBER:          134:247252
TITLE:                    Use of pentagastrin to inhibit gastric acid secretion
                          or as a diuretic
INVENTOR(S):              Pisegna, Joseph R.; Wank, Stephen
PATENT ASSIGNEE(S):       The Regents of the University of California, USA
SOURCE:                   PCT Int. Appl., 42 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
```

WO 2001022985 A1 20010405 WO 2000-US26992 20000928
WO 2001022985 C2 20020926
W: CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1999-156491P P 19990928
US 2000-671764 A 20000927
AB Pentagastrin, when administered in conjunction with a proton pump inhibitor (PPI), is synergistic with the PPI and significantly increases the efficacy of the PPI in reducing/mitigating excess gastric acid secretion.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:868315 CAPLUS
DOCUMENT NUMBER: 135:55833
TITLE: Rabeprazole for the prevention of pathologic and symptomatic relapse of erosive or ulcerative gastroesophageal reflux disease
AUTHOR(S): Caos, Antonio; Moskovitz, Morry; Dayal, Yogeshwar; Perdomo, Carlos; Niecestro, Robert; Barth, Jay
CORPORATE SOURCE: Rabeprazole Study Group, Central Florida Clinical Studies, Ocoee, FL, USA
SOURCE: American Journal of Gastroenterology (2000), 95(11), 3081-3088
CODEN: AJGAAR; ISSN: 0002-9270
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We evaluated the effectiveness and safety profile of 10 and 20 mg of rabeprazole, a new proton pump inhibitor, once daily vs. placebo in preventing endoscopic and symptomatic relapse for up to 1 yr among patients with healed erosive or ulcerative gastroesophageal reflux disease (GERD). The 52-wk trial used a multicenter, randomized, double-blind, parallel-group design in which 209 men and women were assigned to 10 or 20 mg of rabeprazole once daily in the morning or placebo. Both rabeprazole doses were significantly superior to placebo in preventing endoscopic relapse ($p < 0.001$), and 20 mg was significantly more effective than 10 mg ($p < 0.04$). Both doses were also significantly superior to placebo in reducing the frequency and severity of heartburn relapse ($p < 0.001$). When adjusted for differences in exposure to study medication, no significant differences were found in the incidence of adverse events. No clin. significant changes were found regarding clin. lab. parameters, vital signs, electrocardiograms, ophthalmol. evaluations, body wt., serum gastrin, and enterochromaffin-like cell histol. Once-daily therapy with 10 or 20 mg of rabeprazole effectively prevents pathol. and symptomatic GERD relapse. The 20-mg dose is significantly more effective than the 10-mg dose in preventing endoscopic recurrence. Treatment was well tolerated, and no clin. significant safety findings emerged. Our findings support rabeprazole's efficacy in preventing GERD recurrence with excellent tolerability and a short-term favorable safety profile.
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:717677 CAPLUS
DOCUMENT NUMBER: 134:231738
TITLE: Initial potency of lansoprazole and omeprazole tablets on pentagastrin-stimulated gastric acid secretion -- a placebo-controlled study in healthy volunteers
AUTHOR(S): Muller, P.; Goksu, M. A.; Fuchs, W.; Schluter, F.; Simon, B.
CORPORATE SOURCE: Krankenhaus Salem, Heidelberg, D-69120, Germany
SOURCE: Alimentary Pharmacology and Therapeutics (2000), 14(9), 1225-1229
CODEN: APTHEN; ISSN: 0269-2813
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of lansoprazole and omeprazole tablets on pentagastrin-stimulated acid secretion were compared in Helicobacter pylori-neg.

healthy male volunteers. [redacted]stric acid response to submaximal pentagastrin stimulation (0.6 .mu.g/h/k[redacted] was detd. 12.5-14.5 h after ad[redacted]istration of the test drugs. Lansoprazole 15-mg and 30-mg as well as omeprazole 20-mg tablets caused a marked decrease in gastric acid secretion, showing equipotency for the 15-mg lansoprazole and 20-mg omeprazole tablets. Their efficacy, however, was lower than that of 30-mg lansoprazole tablets. In addn., the interindividual variation after omeprazole tablets was higher than that following lansoprazole. Neither 7.5-mg lansoprazole nor 10-mg omeprazole tablets were clearly different from placebo in their effects on the 1st 2 days. The drugs were well tolerated. No clin. relevant influence was found on either lab. screening or cardiovascular parameters. Thus, lansoprazole 15-30-mg tablets produce a stronger acid inhibition and a lower interindividual variability than the new omeprazole 20-mg tablets on days 1 and 2 of administration.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:510647 CAPLUS

DOCUMENT NUMBER: 133:232620

TITLE: Rabeprazole produces rapid, potent, and long-acting inhibition of gastric acid secretion in subjects with Helicobacter pylori infection

AUTHOR(S): Ohning, G. V.; Barbuti, R. C.; Kovacs, T. O. G.; Sytnik, B.; Humphries, T. J.; Walsh, J. H.

CORPORATE SOURCE: Division of Digestive Diseases, Department of Medicine, CURE/UCLA/Digestive Diseases Research Center, Glahs, VA, USA

SOURCE: Alimentary Pharmacology and Therapeutics (2000), 14(6), 701-708
CODEN: APTHEN; ISSN: 0269-2813

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The acid inhibiting activity and duration of action of different doses of rabeprazole, a substituted benzimidazole characterized as a highly potent and irreversible H+,K+-ATPASE inhibitor, were compared in subjects infected with Helicobacter pylori. A total of 38 subjects (mean age 39.3 yr) were enrolled in a single-center, double-blind, randomized, crossover study. All subjects were confirmed pos. for H. pylori by 14C urea breath test and ELISA serologies. Subjects were divided into two groups of 19 to receive two doses of rabeprazole, either 5 and 20 mg or 10 and 40 mg, and placebo, given in random order daily in the morning for 7 days. Peptone-stimulated acid, pH, and gastrin measurements were made for 24 h after the 1st dose and for 48 h after the 7th dose. Peptone-stimulated acid secretion rates were decreased from 12.5 to 6.7, 4.0, 1.5, and 0.26 h after initial 5, 10, 20, and 40 mg doses, resp.; to 7.3, 4.3, 2.1, and 1.2 mmol/h 23 h after the initial dose; and to 2.4, 2.6, 0.6, and 0.8 mmol/h 23 h after the 7th dose. After 48 h, stimulated acid secretion had recovered less than 40% for all treatment groups compared to placebo. Median intragastric pH also increased from 2.0 with placebo to 4.9, 6.2, 6.6 and 6.9 during the 24-h period after the 7th dose of 5, 10, 20, and 40 mg. The 20 mg dose of rabeprazole produced equiv. acid inhibition to the 40 mg dose with less increase in plasma gastrin. Rabeprazole in doses from 5 to 40 mg was a highly effective inhibitor of gastric acid secretion in subjects infected with H. pylori. The inhibition was rapid, dose-related, and long-acting, with less than 50% recovery of acid by 48 h after the 7th dose. The optimal acid ID in these subjects appeared to be 20 mg daily, however 5 mg and 10 mg doses produced potent inhibition of gastric acid secretion.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 6 OF 11 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 1998365261 MEDLINE

DOCUMENT NUMBER: 98365261 PubMed ID: 9701531

TITLE: An ascending single-dose safety and tolerance study of an oral formulation of rabeprazole (E3810).

AUTHOR: Lew E A; Barbuti R C; Kovacs T O; Sytnic B; Humphries T J; Walsh J H

CORPORATE SOURCE: CURE/UCLA/Digestive Disease Research Center, Department of Medicine, West Los Angeles Veterans Administration Medical

Center, Los [redacted]ngeles, CA 90073, USA.
SOURCE: ALIMENTARY [redacted]ARMACOLOGY AND THERAPEUTICS, (1[redacted]3 Jul) 12 (7) 667-72.
Journal code: 8707234. ISSN: 0269-2813.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981029
Last Updated on STN: 19981029
Entered Medline: 19981020

AB BACKGROUND: Proton pump inhibitors such as omeprazole produce a long-lasting inhibition of ***gastric*** ***acid*** ***secretion*** associated with significant increases in plasma ***gastrin*** . ***Rabeprazole*** (E3810) is a new substituted benzimidazole H+,K+ ATPase inhibitor. It acts as an irreversible, non-competitive inhibitor of the H+,K+ ATPase and preliminary studies demonstrate that rabeprazole produces a potent and long-lasting inhibition of ***gastric*** ***acid*** ***secretion*** and a low level of hypergastrinaemia. AIM: This randomized, double-blind, placebo-controlled study was performed to further examine the effects of different single doses of rabeprazole on ***gastric*** ***acid*** ***secretion*** and serum gastrin. METHODS: In this study, four groups of 10 healthy, non-smoking Helicobacter pylori-negative men (mean age 22.5 +/- 3.9 years) received single oral doses of 10, 20, 30 and 40 mg of rabeprazole. Two of the 10 volunteers in each group received placebo as part of the double-blind study design. All volunteers who entered into the study had a normal gastric acid secretory capacity as evaluated by pentagastrin challenge. Prior to administration of the first dose of test drug, volunteers underwent an inpatient 24-h measurement of baseline intragastric pH. One week later, volunteers received the test drug and again underwent an inpatient 24-h measurement of intragastric pH. During both periods, plasma samples were collected at specified intervals over 48 h and were sent for analysis of rabeprazole and gastrin levels. RESULTS: Administration of rabeprazole resulted in a dose-dependent increase in the duration and extent of intragastric pH elevation. The response among all volunteers receiving drug was significantly different from placebo, with greater acid inhibition occurring in the 30 and 40 mg groups. In addition, there was also a dose-related increase in plasma gastrin. The pharmacokinetics of rabeprazole were similar to those of other proton pump inhibitors with a t1/2 of between 0.7 and 1.0 h. There were no clinically significant effects on patient laboratory tests or serious adverse events. CONCLUSIONS: The results of this study suggest that rabeprazole is as potent as omeprazole and lansoprazole in inhibiting ***gastric*** ***acid*** ***secretion*** .

L11 ANSWER 7 OF 11 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97099391 MEDLINE
DOCUMENT NUMBER: 97099391 PubMed ID: 8943968
TITLE: Duodenogastric reflux causes growth stimulation of foregut mucosa potentiated by gastric acid blockade.
AUTHOR: Wetscher G J; Hinder R A; Kretchmar D; Stinson R; Perdikis G; Smyrk T; Klingler P J; Adrian T E
CORPORATE SOURCE: Department of Surgery, Creighton University, Omaha, Nebraska, USA.
SOURCE: DIGESTIVE DISEASES AND SCIENCES, (1996 Nov) 41 (11) 2166-73.
Journal code: 7902782. ISSN: 0163-2116.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961219

AB We investigated whether duodenogastric reflux (DGR) together with gastroesophageal reflux causes growth stimulation of the foregut mucosa and if additional gastric acid suppression enhances the effect of DGR. DGR

was induced in rats using a split gastroenterostomy. A cardiomyotomy was performed across the gastroesophageal junction in order to enhance reflux into the esophagus. DGR rats were divided into six subgroups: DGR, DGR + truncal vagotomy, DGR + omeprazole, DGR + gastrin receptor blockade, DGR + ***omeprazole*** + ***gastrin*** receptor blockade, and DGR + gastrin. Two sham groups, one with and one without omeprazole treatment, served as controls. DGR significantly increased the weight and DNA content of the esophageal and gastric mucosa, which was further enhanced by vagotomy or omeprazole. Histology revealed foveolar hyperplasia in the stomach and esophageal mucosal hyperplasia in these groups. In addition, severe ***esophagitis*** was found in the DGR group receiving omeprazole. Omeprazole without DGR had no growth-stimulating effect on the foregut mucosa. DGR-induced growth stimulation was accompanied by hypergastrinemia. Increased growth in the stomach but not the esophagus was inhibited by gastrin receptor blockade. Gastrin administration did not result in enhancement of DGR-induced growth stimulation of the foregut mucosa. It is concluded that DGR, often present in severe reflux ***esophagitis*** , causes mucosal growth of the foregut of rats. This trophic response may explain why severe reflux ***esophagitis*** is associated with an increased risk of esophageal adenocarcinoma. DGR-induced growth stimulation of the foregut is potentiated by gastric acid suppression, suggesting that chronic antisecretory medication in gastroesophageal reflux may not always be advisable. Omeprazole + DGR caused severe esophageal damage, which may explain why antisecretory medication may fail to heal severe reflux ***esophagitis*** .

L11 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:485158 CAPLUS
DOCUMENT NUMBER: 115:85158
TITLE: 24-Hour intragastric acidity and plasma gastrin during long-term treatment with omeprazole or ranitidine in patients with reflux esophagitis
AUTHOR(S): Lind, Tore; Cederberg, C.; Idstroem, J. P.; Loenroth, H.; Olbe, L.; Lundell, L.
CORPORATE SOURCE: Dep. Surg., Sahlgren's Hosp., Goeteborg, S-413 45, Swed.
SOURCE: Scandinavian Journal of Gastroenterology (1991), 26(6), 620-6
CODEN: SJGRA4; ISSN: 0036-5521
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The redn. in intragastric acidity and the subsequent increase in plasma gastrin were compared during long-term treatment with either omeprazole or ranitidine in 19 patients with erosive reflux esophagitis. The patients received 40 mg omeprazole in the morning or 300 mg ranitidine twice daily. After healing, half the dose was given as maintenance treatment for 1 yr. Intragastric acidity and plasma gastrin was measured 24 h before entry and monthly with the high dose and after 1, 6, and 12 mo with the low dose. Omeprazole reduced intragastric acidity more effectively than ranitidine ($p < 0.001$). This difference in efficacy was more pronounced during the daytime. Plasma gastrin increased more after omeprazole than after ranitidine ($p < 0.01$), and both drugs showed a normal postprandial response and approached fasting levels before the next dose. During long-term treatment with 20 mg omeprazole in the morning no progressive alterations were obsd. in 24-h intragastric acidity or plasma gastrin.

L11 ANSWER 9 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1992:131854 BIOSIS
DOCUMENT NUMBER: BR42:59554
TITLE: PARIETAL CELL FUNCTIONS IN CULTURE.
AUTHOR(S): CHEW C S
CORPORATE SOURCE: DEP. PHYSIOL., MOREHOUSE SCH. MED., ATLANTA, GA., USA.
SOURCE: 7TH INTERNATIONAL CONFERENCE ON EXPERIMENTAL ULCER, BERLIN, GERMANY, OCTOBER 15-18, 1991. DIGESTION, (1991) 49 (SUPPL 1), 2-3.
CODEN: DIGEBW. ISSN: 0012-2823.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L11 ANSWER 10 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1989:226920 BIOSIS

DOCUMENT NUMBER: BA87:118537
TITLE: RELATIONSHIP BETWEEN REDUCTION OF GASTRIC ACID SECRETION AND PLASMA GASTRIN CONCENTRATION DURING OMEPRAZOLE TREATMENT.
AUTHOR(S): LIND T; CEDERBERG C; FORSSELL H; OLAUSSON M; OLBE L
CORPORATE SOURCE: DEP. SURGERY, SAHLGREN'S HOSP., S-413 45 GOTHENBURG, SWEDEN.
SOURCE: SCAND J GASTROENTEROL, (1988) 23 (10), 1259-1266.
CODEN: SJGRA4. ISSN: 0036-5521.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB We have studied the relationship between reduction of ***gastric*** ***acid*** ***secretion*** and fasting plasma ***gastrin*** ***omeprazole*** for 5 days in daily doses of 5, 10 ,20, 40, or 80 mg. Acid secretion and fasting gastrin concentration were measured 6 h (maximal omeprazole effect) and 24 h (minimal omeprazole effect) after the fifth omeprazole dose. Omeprazole in doses lower than 20 mg daily did not suppress pentagastrin-stimulated acid secretion in all subjects 6 h after dosing on the 5th day. Doses of 20-80 mg omeprazole, however, significantly reduced acid secretion 24 h after the fifth dose, the range being 36-76%. A relationship between degree of acid inhibition and fasting gastrin concentration was observed. However, acid secretion needed to be reduced by more than 80% before gastrin levels were clearly affected. This degree of acid inhibition was only achieved 6 h after administration of omeprazole in doses of 20 mg and higher. The inhibitory effect of omeprazole on acid secretion decreased 24 h after dosing. Thus, fasting gastrin concentrations were moderately increased in the beginning and normalized at the end of each 24-h period during treatment with daily doses of 20-80 mg omeprazole.

L11 ANSWER 11 OF 11 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 88092729 MEDLINE
DOCUMENT NUMBER: 88092729 PubMed ID: 3695537
TITLE: Use of a five-day test to predict the long-term effects of gastric antisecretory agents on serum gastrin in rats.
AUTHOR: Katz L B; Schoof R A; Shriver D A
CORPORATE SOURCE: Research Laboratories, Ortho Pharmaceutical Corporation, Raritan, NJ 08869-0602.
SOURCE: JOURNAL OF PHARMACOLOGICAL METHODS, (1987 Dec) 18 (4) 275-82.
Journal code: 7806596. ISSN: 0160-5402.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198801
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19900305
Entered Medline: 19880126

AB It has been hypothesized that prolonged achlorhydria causes compensatory elevation of serum gastrin, and that there is an association in rats between sustained hypergastrinemia, hyperplasia of gastric enterochromaffin-like cells, and subsequent formation of gastric carcinoids in 2-year carcinogenicity studies. The present study examined whether daily administration of gastric antisecretory drugs in rats for 4 days could cause hypergastrinemia associated with inhibition of acid output. Rats were dosed orally for 4 days with the histamine H2-receptor antagonist ranitidine or the H+,K+-sensitive ATPase inhibitor omeprazole, and examined on day 5 for effects on ***gastric*** ***acid*** ***secretion*** and serum ***gastrin*** . ***Omeprazole*** (138 mg/kg/day significantly inhibited ***gastric*** ***acid*** ***secretion*** and increased serum gastrin levels. Large, single daily doses of ranitidine (1000-2000 mg/kg/day) had no effect on 24-hr acid or gastrin secretion; however, ranitidine did inhibit next-day acid secretion with associated increases in serum gastrin when administered in three divided doses. These results with ranitidine support the hypothesis that a sustained gastric antisecretory action will cause a compensatory hypergastrinemia, regardless of the antisecretory agent used. The ability to detect increased serum gastrin levels associated with inhibition of acid secretion, after administration of antisecretory agents for only 4 days, suggest that this short 5-day test may be useful for determining the potential of antisecretory agents to cause hypergastrinemia due to 24-hr

inhibition of acid secreti and may be predictive of long-term hyperplastic changes.

=> d his

(FILE 'HOME' ENTERED AT 14:44:23 ON 17 NOV 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 14:44:51 ON 17 NOV 2002

```
L1          185 S GASTRIC PROTON PUMP INHIBITOR
L2        36717 S RABEPRAZOLE OR OMEPRAZOLE OR LANSOPRAZOLE OR PANTOPRAZOLE
L3        81692 S GASTRIN OR PENTAGASTRIN
L4         3158 S (L1 OR L2 ) (P) L3
L5        89633 S (GASTRIC ACID SECRETION) OR (ZOLLINGER ELLISON SYNDROME) OR (
L6         1066 S L4 (P) L5
L7          418 DUPLICATE REMOVE L6 (648 DUPLICATES REMOVED)
L8          217 S L7 (P) TREAT?
L9          108 S (L1 OR L2) (A) L3
L10          19 S L9 (P) L5
L11          11 DUPLICATE REMOVE L10 (8 DUPLICATES REMOVED)
```

=> s antibiotic or penicillin or tetracycline or macrolide or cephalosporin or fluoroguinolone

```
L12     969962 ANTIBIOTIC OR PENICILLIN OR TETRACYCLINE OR MACROLIDE OR CEPHALO
               SPORIN OR FLUOROGUINOLONE
```

=> s l8 (p) l12

```
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L55 (P) L77'
L13          9 L8 (P) L12
```

=> s l13 not l11

```
L14          9 L13 NOT L11
```

=> d l14 1-9 ibib abs

L14 ANSWER 1 OF 9 MEDLINE

ACCESSION NUMBER: 2000329062 MEDLINE
DOCUMENT NUMBER: 20329062 PubMed ID: 10872661
TITLE: Parietal cell protrusions and fundic gland cysts during omeprazole maintenance treatment.
COMMENT: Comment in: Hum Pathol. 2000 Dec;31(12):1536-7
AUTHOR: Cats A; Schenk B E; Bloemena E; Roosedaal R; Lindeman J; Biemond I; Klinkenberg-Knol E C; Meuwissen S G; Kuipers E J
CORPORATE SOURCE: Department of Gastroenterology, Academic Hospital Vrije Universiteit, Amsterdam, The Netherlands.
SOURCE: HUMAN PATHOLOGY, (2000 Jun) 31 (6) 684-90.
Journal code: 9421547. ISSN: 0046-8177.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000714
Last Updated on STN: 20020214
Entered Medline: 20000706

AB Parietal cell protrusion (PCP), swelling and bulging of parietal cells, has been observed in the oxyntic mucosa of patients receiving ***omeprazole*** . The frequency of this event and the underlying mechanisms remain to be clarified. As such, it is unknown whether there is a relation with either serum ***gastrin*** or Helicobacter pylori infection, and whether PCP predisposes to the development of fundic gland cysts (FGC). We therefore investigated the development of PCP and FGC in ***gastroesophageal*** ***reflux*** ***disease*** (GERD) patients ***treated*** with ***omeprazole*** and correlated findings to duration of therapy, ***gastrin*** , and H pylori infection. In a randomized, double-blinded study, GERD patients were evaluated by endoscopy with biopsy sampling for histology and culture at baseline, and after 3 and 12 months' therapy with ***omeprazole*** 40

mg daily. H pylori-positive patients were randomized to additional eradication therapy or placebo ***antibiotics*** at baseline. All histological slides were scored blinded for time and outcome of culture for the presence of PCP and FGC. Fasting serum samples from all visits were used for ***gastrin*** measurements. The prevalence of PCP increased during ***omeprazole*** therapy from 18% at baseline to 79% and 86% at 3 and 12 months (P < .001, baseline v both 3 and 12 months). The prevalence of FGC increased from 8% to 17% and 35% (P < .05, baseline v 12 months). The prevalence of PCP and FGC did not differ among the H pylori-positive and H pylori-negative patients at baseline (PCP 16% v 20% and FGC 7% v 8%, respectively). Whereas H pylori eradication did not significantly affect development of PCP (P = .7), FGC developed significantly more often in the H pylori-eradicated patients when compared with persistent H pylori-positive patients (P < .05). PCP development was related to serum ***gastrin*** rise during therapy. In conclusion, PCP occurs in most patients within the first months of ***omeprazole*** ***treatment*** and is related to increased ***gastrin*** levels. FGC develops more gradually and is enhanced by H pylori eradication.

L14 ANSWER 2 OF 9 MEDLINE
ACCESSION NUMBER: 2000158105 MEDLINE
DOCUMENT NUMBER: 20158105 PubMed ID: 10695558
TITLE: Helicobacter pylori and its eradication in rosacea.
AUTHOR: Szlachcic A; Sliwowski Z; Karczewska E; Bielanski W; Pytko-Polonczyk J; Konturek S J
CORPORATE SOURCE: Department of Physiology, University School of Medicine, Cracow, Poland.
SOURCE: JOURNAL OF PHYSIOLOGY AND PHARMACOLOGY, (1999 Dec) 50 (5) 777-86.
Journal code: 9114501. ISSN: 0867-5910.
PUB. COUNTRY: Poland
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000407
Last Updated on STN: 20000407
Entered Medline: 20000328

AB Rosacea is a common condition of unknown etiology usually accompanied by gastrointestinal symptoms and favorably responding to the ***treatment*** with ***antibiotics*** . This study was designed to examine the prevalence of gastric Helicobacter pylori (Hp) infection verified by 13C-UTB-test, CLO, Hp culture and serology (IgG) in patients with rosacea. Gastroduodenoscopy was combined with ***pentagastrin*** secretory test and antral and fundic biopsy samples were taken for histological evaluation (the Sydney system). Blood samples were also taken for the determination of plasma ***gastrin*** using RIA and plasma interleukin (IL)-8 and tumor necrosis factor alpha (TNFalpha) using ELISA. This study was performed in 60 patients, 31-72 year old, with visible papules and pustules associated with erythema and flushing on the face and on 60 age- and gender-matched patients without any skin diseases but with similar as in rosacea gastrointestinal symptoms but without endoscopic changes in gastroduodenal mucosa (non-ulcer dyspepsia - NUD). The Hp prevalence in rosacea patients was about 88 % as compared to 65% in control NUD patients. Among rosacea patients, 67% were cytotoxin associated gene A (CagA) positive, while in NUD patients only 32% were CagA positive. Rosacea patients showed gastritis with activity of about 2.1 in antrum and 0.9 in the corpus of the stomach while those with NUD only mild gastritis with activity of approximately 1.0) confined to the antrum only. Following initial examination, typical 1 wk anti-Hp therapy including ***omeprazole*** (20 mg bd.), clarithromycin (500 mg bd.) and metronidazol (500 mg bd.) was carried out. After eradication, 51 out of 53 ***treated*** rosacea patients became Hp negative. Within 2-4 weeks, the symptoms of rosacea disappeared in 51 patients, markedly declined in 1 and remained unchanged in 1 other subject. A dramatic reduction in activity of gastritis (to 0.3 in antrum and to 0.1 in corpus) was observed. Basal plasma ***gastrin*** decreased from 48 +/- 5 pM before to 17+/-3 pM after eradication, while ***pentagastrin*** -induced maximal (MAO) declined, respectively, from about 16.6 +/- 4.2 to 8.5 +/- 1.8 mmol/h. Plasma TNFalpha and IL-8 were reduced after the therapy by 72% and 65%, respectively. We conclude that: 1) Rosacea is a

disorder with various gastrointestinal symptoms closely related to gastritis, especially involving the antrum mucosa, with Hp expressing cagA in the majority of cases and elevated plasma levels of TNFalpha and IL-8; 2) The eradication of Hp leads to a dramatic improvement of symptoms of rosacea and reduction in related gastrointestinal symptoms, gastritis, hypergastrinemia and ***gastric*** ***acid*** ***secretion*** ; and 3) Rosacea could be considered as one of the major extragastric symptoms of Hp infection probably mediated by Hp-related cytotoxins and cytokines.

L14 ANSWER 3 OF 9 MEDLINE
ACCESSION NUMBER: 1999292020 MEDLINE
DOCUMENT NUMBER: 99292020 PubMed ID: 10365898
TITLE: H+/K+-adenosine triphosphatase mRNA in gastric fundic gland mucosa in patients infected with Helicobacter pylori.
AUTHOR: Furuta T; Baba S; Takashima M; Shirai N; Xiao F; Futami H; Arai H; Hanai H; Kaneko E
CORPORATE SOURCE: First Dept. of Medicine, Hamamatsu University School of Medicine, Japan.
SOURCE: SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY, (1999 Apr) 34 (4) 384-90.
Journal code: 0060105. ISSN: 0036-5521.
PUB. COUNTRY: Norway
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990730
Last Updated on STN: 19990730
Entered Medline: 19990722

AB BACKGROUND: How Helicobacter pylori infection affects ***gastric*** ***acid*** ***secretion*** has not been made clear. This study aimed to elucidate the effects of H. pylori infection on H+/K+-adenosine triphosphatase (ATPase) mRNA in gastric fundic gland mucosa. METHODS: Twenty patients with chronic gastritis and H. pylori infection were ***treated*** with ***lansoprazole*** and ***antibiotics*** . Before and 1 month after ***treatment*** gastroduodenoscopy was performed, and changes in the amount of H+/K+-ATPase mRNA in the fundic gland mucosa, gastric juice pH, and serum ***gastrin*** levels were determined. RESULTS: The amount of H+/ K+-ATPase mRNA in the fundic gland mucosa was increased in patients with eradication of H. pylori, in whom significant decreases in gastric juice pH and serum ***gastrin*** levels were observed. No significant changes were observed in patients without eradication of H. pylori. CONCLUSIONS: These results suggest that one of the mechanisms by which H. pylori infection suppresses acid secretion is by the inhibition of proton pump synthesis in parietal cells.

L14 ANSWER 4 OF 9 MEDLINE
ACCESSION NUMBER: 95036730 MEDLINE
DOCUMENT NUMBER: 95036730 PubMed ID: 7949462
TITLE: Treatment of peptic ulcers from now to the millennium.
AUTHOR: Pounder R E
CORPORATE SOURCE: Royal Free Hospital and School of Medicine, London, UK.
SOURCE: BAILLIERES CLINICAL GASTROENTEROLOGY, (1994 Jun) 8 (2) 339-50. Ref: 61
Journal code: 8704786. ISSN: 0950-3528.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19950110
Entered Medline: 19941208

AB The present strategies for the management of peptic ulceration are well tolerated and clinically effective. Histamine H2-receptor antagonists can be used for mild to moderate disease, and proton pump inhibitors are of particular benefit for patients with severe peptic ulceration and the ***Zollinger*** - ***Ellison*** ***syndrome*** . However, none of these ***treatments*** provides protection against recurrent

ulceration, except when taken as long-term continuous ***treatment*** . Long-term exposure to pharmacological agents raises problems of safety, particularly relating to a lack of intragastric acidity. In addition, the accelerated development of ***atrophic*** ***gastritis*** in patients receiving ***omeprazole*** requires investigation and assessment. It is unlikely that there will be any major development in the area of control of ***gastric*** ***acid*** ***secretion*** , except perhaps the introduction of specific immunization against ***gastrin*** . However, the clinical benefit of this strategy awaits assessment. The main area for development must be the introduction of convenient and effective regimens for the eradication of Helicobacter pylori infection. Existing regimens are either simpler and relatively ineffective, or too complicated for widespread application. Bearing in mind the long gestation period of any new drug, it seems likely that the only innovative drug that will be introduced for the management of peptic ulceration before the millennium will be ranitidine bismuth citrate, an antisecretory anti-H. pylori drug that will usually be used in combination with an ***antibiotic*** .

L14 ANSWER 5 OF 9 MEDLINE
ACCESSION NUMBER: 94254616 MEDLINE
DOCUMENT NUMBER: 94254616 PubMed ID: 8196467
TITLE: [Lansoprazole--profile of a new proton pump inhibitor]. Lansoprazol--Profil eines neuen Protonenpumpenhemmers.
AUTHOR: Seifert E
CORPORATE SOURCE: I. Med. Klinik, Stadt. Krankenhaus Kemperhof Koblenz.
SOURCE: LEBER, MAGEN, DARM, (1994 Mar) 24 (2) 66-8, 71. Ref: 27
Journal code: 0311747. ISSN: 0300-8622.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199406
ENTRY DATE: Entered STN: 19940707
Last Updated on STN: 19940707
Entered Medline: 19940627
AB ***Lansoprazole*** , a new proton pump inhibitor, selectively inhibits the H+/K(+)-ATPase. Its inhibitory effect on basal and ***gastrin*** stimulated ***gastric*** ***acid*** ***secretion*** is equal to ***omeprazole*** and stronger than that of H2-receptor antagonists. Healing rates concerning gastric and duodenal ulcers and refluxesophagitis are significantly higher compared to H2-receptor antagonists and at least comparable to ***omeprazole*** . Regarding pilot studies in H. pylori eradication therapy, ***lansoprazole*** in combination with various ***antibiotics*** is expected to show good eradication rates. Considering its excellent safety and interaction profile ***lansoprazole*** is effective and safe in ***treating*** acid related disorders.

L14 ANSWER 6 OF 9 MEDLINE
ACCESSION NUMBER: 93117994 MEDLINE
DOCUMENT NUMBER: 93117994 PubMed ID: 1475769
TITLE: [Diagnosis of peptic ulcer disease]. Diagnostik beim peptischen Ulkusleiden.
AUTHOR: Sheurer U; Merki H
CORPORATE SOURCE: Abteilung fur Gastroenterologie, Medizinischen Klinik, Inselspatal, Bern.
SOURCE: THERAPEUTISCHE UMSCHAU, (1992 Nov) 49 (11) 735-42. Ref: 30
Journal code: 0407224. ISSN: 0040-5930.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199302
ENTRY DATE: Entered STN: 19930219
Last Updated on STN: 19930219
Entered Medline: 19930204
AB Today the upper gastrointestinal endoscopy is the diagnostic tool of

choice to detect peptic ga[redacted]oduodenal lesions. In case of substantial gastric outlet obstruction[redacted] strong suspicion of perforate[redacted]lcer, an upper gi-transit with barium or water soluble contrast medium in suspected perforated ulcers may be useful. Gastric ulcers are endoscopically controlled up to their complete healing and biopsies taken at each endoscopy in order to rule out gastric cancer. In contrast, duodenal ulcers are rarely malignant and uncomplicated duodenal ulcers, correctly ***treated*** with ***omeprazole*** over 8 weeks do not necessarily need a final endoscopic control. Since about 5% of duodenal ulcers ***treated*** with H2 blockers or mucosal protective agents do not heal within 8 weeks however, an endoscopic control of the healing is recommended. In peptic ulcer patients tests for detection of helicobacter pylori are only needed in presence of a hard indication for immediate eradication: Frequent ulcer recurrencies, complicated ulcer disease or very painful ulcer relapses, because the eradication therapy is often not well tolerated and the patient compliance therefore compromised. 30% of helicobacter infected patients have ***antibiotic*** resistant strains and there is no sufficient longterm experience with the eradication therapy available (4) to 8 weeks after ***treatment*** of the helicobacter pylori infection the effect on ulcer healing and infection should be verified. Determinations of plasma ***gastrin*** levels in peptic ulcer patients are mandatory in patients with suspected ***Zollinger*** - ***Ellison*** ***syndrome*** or patients with ***treatment*** resistant ulcers or recurrent ulcers after vagotomy or partial gastric resection.(ABSTRACT TRUNCATED AT 250 WORDS)

L14 ANSWER 7 OF 9 MEDLINE
ACCESSION NUMBER: 93012546 MEDLINE
DOCUMENT NUMBER: 93012546 PubMed ID: 1397740
TITLE: Helicobacter pylori, peptic ulcer disease and inhibition of gastric acid secretion.
AUTHOR: Rune S
CORPORATE SOURCE: Department of Gastroenterology, Glostrup Hospital, Denmark.
SOURCE: DIGESTION, (1992) 51 Suppl 1 11-6. Ref: 20
Journal code: 0150472. ISSN: 0012-2823.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW LITERATURE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199211
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19950206
Entered Medline: 19921125
AB Recent studies have been reviewed to establish the possible importance of the interaction between Helicobacter pylori infection and ***gastric*** ***acid*** ***secretion*** . H. pylori infection results in increased ***gastrin*** release, but this does not lead to gastric acid hypersecretion and ***gastrin*** normalizes after eradication of the infection. An optimal, well-tolerated ***treatment*** strategy against H. pylori infection has not yet been clearly defined. One potentially useful approach may be to improve the antibacterial efficacy of ***antibiotics*** by effectively regulating gastric acidity. H2-receptor antagonists have no effect against H. pylori infection, while ***omeprazole*** (an acid pump inhibitor) appears to have a bacteriostatic action. Combination therapy with ***omeprazole*** and amoxycillin has been found to eradicate H. pylori in 50-80% of patients with duodenal ulcer, leading to a significant reduction in ulcer recurrence.

L14 ANSWER 8 OF 9 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 95325000 EMBASE
DOCUMENT NUMBER: 1995325000
TITLE: Eradication of Helicobacter pylori.
AUTHOR: Harris A.W.; Misiewicz J.J.
CORPORATE SOURCE: Dept Gastroenterology and Nutrition, Central Middlesex Hospital, Acton Lane,London NW10 7NS, United Kingdom
SOURCE: Bailliere's Clinical Gastroenterology, (1995) 9/3 (583-613).
ISSN: 0950-3528 CODEN: BCGAER
COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 004 Microbiology
037 Drug Literature Index
038 Adverse Reactions Titles
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Although there are numerous publications reporting eradication results, the general picture is confused by the bewildering multiplicity of ***treatment*** schedules employed by the various workers. The over-riding need now is for large scale trials, and more especially for direct comparisons of different ***treatment*** regimens in the same populations of patients. Such data are entirely absent from the literature at present. Standardization of definitions and of methodology pertaining to diagnosis of eradication, recording of side effects, measurement of compliance and determination of recurrence or of reinfection, is badly needed. As the definition of eradication remains arbitrary, it is important to include genome fingerprinting techniques in the long-term follow-up for recurrence, so that the question of reinfection versus recrudescence can be examined. Because of the wide differences in the agents used in H. pylori eradication therapies, proper double-blinding of ***treatment*** trials remains a difficult problem. This can be dealt with to some extent by ensuring that the interpretation of tests for H. pylori eradication is performed by personnel unaware of the clinical details. Review of the existing data on eradication of H. pylori indicates that clinically useful results can be achieved in some 70 to 95% of patients, on an intention to ***treat*** basis. Compliance, side effects and resistance to metronidazole remain the limiting factors. Efficacy, freedom from side effects, simplicity and low cost will determine the success of any regimen in the future. At present, it is not possible to make firm recommendations in favour of one regimen over another, but it seems reasonable to forecast that dual therapies consisting of a PPI and an ***antibiotic*** will receive much attention. Preparations consisting of an H2RA associated with a bismuth compound, which are used together with an ***antibiotic*** are an interesting approach. Compliance should be as good as with a normal dual therapy and the eradication results look promising. The advantages of dual therapies that include a PPI lie in their simplicity, in not relying on imidazole for their anti-H. pylori effect but on the profound inhibition of acid output produced by the PPI. Thus PPI based dual therapy can probably evoke better compliance than the more complicated regimens. The use of PPIs has other advantages in addition to decreasing the MIC90 of the ***antibiotic*** combined with it. This is because administration of a powerful inhibitor of ***gastric*** ***acid*** ***secretion*** , such as a PPI, will aid the rapid healing of an ulcer crater and will rapidly relieve the symptoms of peptic ulceration. ***Gastrin*** releasing peptide-stimulated acid secretion is raised in duodenal ulcer patient's to approximately sixfold over control levels according to El-Omar et al, and although it returns to normal following the eradication of H. pylori, this process takes time to become effective. Suppression of acid output provides an immediate therapeutic shield, while the decrease in inflammation and acid output secondary to H. pylori eradication can be established. The most widespread resistance to ***antibiotics*** exhibited by H. pylon Is with respect to imidazoles. The prevalence of metronidazole resistance is widespread in the emergent countries, but it is also appreciable in the West, especially in women, who may have been given metronidazole in the ***treatment*** of pelvic infections. Moreover, H. pylori becomes resistant to metronidazole very easily and often as a result of ***treatment*** which includes an imidazole compound. On the other hand, H. pylori resistance to ***macrolides*** is not widespread and does not develop easily during their administration. It is difficult to forecast which ***antibiotic*** will be the most widely used agent in combination with a PPI. Amoxycillin seems quite effective when combined with a PPI administered twice daily, while clarithromycin leads the ***macrolides*** in its in vitro anti-H. pylori activity. Bismuth-based triple regimens have the advantage of familiarity. Ensuring compliance is the duty of the physician initiating the ***treatment*** . The incidence of eradication with these regimens differs in different centres and the reasons for these discrepancies need to be investigated. One week, low dose triple therapy with ***omeprazole*** , clarithromycin and tinidazole or metronidazole appears highly effective, with few side effects and good compliance.

However, data is not available on the pretreatment sensitivity to nitroimidazoles of H. pylori from the patients studied, or on the development of resistant strains during ***treatment*** .

L14 ANSWER 9 OF 9 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 95091873 EMBASE
DOCUMENT NUMBER: 1995091873
TITLE: [Omeprazole and the new proton pump inhibitors].
OMEPRAZOL UND DIE NEUEN PROTONEN PUMPENHEMMER.
AUTHOR: Born P.; Classen M.
CORPORATE SOURCE: II. Medizinische Klinik, Klinikum Rechts der Isar, Technische Universitat, Ismaninger Strasse 22,D-81675 Munchen, Germany
SOURCE: Verdauungskrankheiten, (1995) 13/1 (23-31).
ISSN: 0174-738X CODEN: VERDEJ
COUNTRY: Germany
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 048 Gastroenterology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: German
SUMMARY LANGUAGE: German; English
AB ***Omeprazole*** and the new proton pump inhibitors ***lansoprazole*** and ***pantoprazole*** , specific inhibitors of the H+/K+-ATPase in the parietal cells of the stomach suppress the ***gastric*** ***acid*** ***secretion*** in a way not reached before. Therefore, they are superior to H2-antagonists in the therapy of peptic lesions like reflux oesophagitis, duodenal ulcer and ***Zollinger*** - ***Ellison*** ***syndrome*** . Although the importance of elevated levels of ***gastrin*** and the possible development of carcinoids is not definitively cleared, long-term ***treatment*** seems to be possible and should be able to prevent surgical intervention in special cases. Special importance proton pump inhibitors get in a combination therapy with ***antibiotics*** to eradicate helicobacter pylori.

=> d his

(FILE 'HOME' ENTERED AT 14:44:23 ON 17 NOV 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 14:44:51 ON 17 NOV 2002
L1 185 S GASTRIC PROTON PUMP INHIBITOR
L2 36717 S RABEPRAZOLE OR OMEPRAZOLE OR LANSOPRAZOLE OR PANTOPRAZOLE
L3 81692 S GASTRIN OR PENTAGASTRIN
L4 3158 S (L1 OR L2) (P) L3
L5 89633 S (GASTRIC ACID SECRETION) OR (ZOLLINGER ELLISON SYNDROME) OR (
L6 1066 S L4 (P) L5
L7 418 DUPLICATE REMOVE L6 (648 DUPLICATES REMOVED)
L8 217 S L7 (P) TREAT?
L9 108 S (L1 OR L2) (A) L3
L10 19 S L9 (P) L5
L11 11 DUPLICATE REMOVE L10 (8 DUPLICATES REMOVED)
L12 969962 S ANTIBIOTIC OR PENICILLIN OR TETRACYCLINE OR MACROLIDE OR CEPH
L13 9 S L8 (P) L12
L14 9 S L13 NOT L11

=> s l7 (p) kit
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L109 (P) KIT'
L15 0 L7 (P) KIT

=> d his

(FILE 'HOME' ENTERED AT 14:44:23 ON 17 NOV 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 14:44:51 ON 17 NOV 2002
L1 185 S GASTRIC PROTON PUMP INHIBITOR
L2 36717 S RABEPRAZOLE OR OMEPRAZOLE OR LANSOPRAZOLE OR PANTOPRAZOLE
L3 81692 S GASTRIN OR PENTAGASTRIN

```
L4          3158 S (L1 OR L2 ) (  L3
L5         89633 S (GASTRIC ACID  ECRETION) OR (ZOLLINGER ELLISON  NDROME) OR (
L6          1066 S L4 (P) L5
L7           418 DUPLICATE REMOVE L6 (648 DUPLICATES REMOVED)
L8           217 S L7 (P) TREAT?
L9           108 S (L1 OR L2) (A) L3
L10           19 S L9 (P) L5
L11           11 DUPLICATE REMOVE L10 (8 DUPLICATES REMOVED)
L12       969962 S ANTIBIOTIC OR PENICILLIN OR TETRACYCLINE OR MACROLIDE OR CEPH
L13            9 S L8 (P) L12
L14            9 S L13 NOT L11
L15            0 S L7 (P) KIT

=> log y
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                      ENTRY    SESSION
FULL ESTIMATED COST                                   98.71      98.92

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                      ENTRY    SESSION
CA SUBSCRIBER PRICE                                   -3.72      -3.72

STN INTERNATIONAL LOGOFF AT 14:55:34 ON 17 NOV 2002
```